Journal of Forensic and Legal Medicine 17 (2010) 1–7

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/jflm

Review

Deaths in custody: Are some due to electronic control devices (including TASER® devices) or excited delirium?

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ARTICLE INFO

Article history:
Received 26 October 2007
Received in revised form 11 March 2008
Accepted 18 May 2008
Available online 9 August 2008

Keywords:
Electronic control device
Electric shocking device
TASER
Excited delirium
Electric stimulation
Conducted energy weapon

ABSTRACT

Deaths have occurred after law-enforcement incidents involving applications of electronic control devices (ECDs) (including TASER® devices). An "excited delirium" syndrome (reported in the literature prior to the development of ECDs currently in use), however, includes several factors that may be related to such deaths in custody. In this review, potential detrimental effects of ECDs are compared with possible changes due to excited delirium. Although extreme (i.e., long-duration or repeated) exposures to ECDs can result in significant hyperkalaemia, acidaemia, and myoglobinemia in animal models, limited applications (such as those normally used in law-enforcement situations) would appear to have only transient effects. In addition, the hyperthermia observed in patients with excited delirium does not seem to be directly exacerbated by ECD applications. ECD use is unlikely to be a common cause of ventricular fibrillation, but other events that are generally associated with excited delirium (e.g., drug use) may be related to subsequent ventricular fibrillation or asystole. Metabolic or respiratory acidosis may only be serious consequences of long-duration or repeated ECD applications. On the basis of current available information, factors other than ECDs themselves may be more important when death occurs after the use of ECDs.

1. Introduction

An "electronic control device" (ECD) is an operator-controlled apparatus that transmits short electric pulses either through electrodes (from a "stun gun," as described by Schmiederer et al.),[1] or through wires attached to darts (e.g., from a TASER® ["Thomas A. Swift's Electric Rifle"] ECD [alternatively referred to as an "electric shocking device" or "conducted energy weapon"]) into a subject, causing involuntary muscle contractions that incapacitate. (TASER® is a registered trademark of TASER International, Inc., Scottsdale, Arizona, USA). Denk et al.[2] suggested that TASER ECDs can be more effective than other models of similar devices. (For a recent comprehensive review of ECD effects, see Jenkinson et al.[3]). Tracings of typical waveforms produced by the TASER model "X26" ECD have been presented previously.[4,5]

Although the muscle contraction caused by ECD application is not synonymous with conventional muscular exercise, there may be some similarities. Rhabdomyolysis (breakdown of muscle tissue to the extent that contents are liberated into the circulation) may be caused by either excessive muscular activity[6] or electrical injury.[7] (Other causes are hyperthermia and direct myotoxins.[8] One may expect any possible electrical injury due to ECD exposure, however, to be quite different from conventional electrical injury (due to, e.g., 60 Hz alternating current), since electrical properties (most importantly, current flow) of the two modalities are dissimilar. An important point is the amount of power delivered in a given period of time, or energy in joules (watt–seconds) (see Fechner et al.[9] for discussion of these factors).

Medical examiners have listed ECDs as a cause of death (see Remsberg [10] for discussion), despite the lack of a clear-cut unifying pathophysiological hypothesis. At the same time, a syndrome often referred to as "excited delirium" (at least in the United States, if not in other countries)[11] is one possible explanation of unanticipated deaths of suspects in police custody.[12] Ross[13] has reviewed factors that have been associated with past cases of deaths in custody presumed to be related to excited delirium.

In news-media accounts of deaths during incidents involving applications of ECDs, the initial focus is often on the devices themselves as the important factor relating to death, with other issues considered to be secondary. The excited delirium syndrome, however, was reported in the literature prior to the development of ECDs currently in use.

The purposes of this paper are to (1) review potential detrimental effects (as reflected in blood factors and physiological measurements) of exposure to ECDs, (2) assess the relative importance of such factors to survival (in comparison with certain aspects of excited delirium), and (3) summarize current opinions regarding the potential role of ECDs versus that of excited delirium during deaths in custody.

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doi:10.1016/j.jflm.2008.05.011

2. Terminology and definitions of excited delirium

Wetli et al.[14] and Karch[15] have listed four components to be included in a definition of the excited delirium syndrome: delirium with agitation, respiratory arrest, hyperthermia, and death. One of the first uses of the specific term "excited delirium" was by Wetli and Fishbain[16] (although "cocaine-induced delirium" had been described before that time). That particular first use of the term was descriptive of behavior, rather than a diagnosis. The syndrome was earlier described under various different terms, including "lethal catatonia".[17] Although death would appear to be required for the definition, Manojlovic et al.[18] have referred to "characteristics of excited delirium" in cases in which the subject survives. Excited delirium may be characterized by a history of either abuse of stimulants or of endogenous mental disease.[19] Other medical conditions with symptoms that may appear similar to those of excited delirium include thyroid storm and other endocrinopathies.[20,21] Wetli[22] noted, "The term agitated or excited delirium is used for the same syndrome, regardless of etiology (i.e., infectious, toxic, pharmacologic, or psychiatric)."

Paquette[23] and Wetli[24] have discussed the controversy over the recognition of excited delirium as a clinical entity. The latest international classification of diseases (ICD) from the world health organization (WHO)[25] (and a modified version of causes of death formatted for forensic medicine[26] does not include the specific term "excited delirium." Some deaths can be classified under the ICD code as "deaths by legal intervention" (including use-of-force by police)[27]. The WHO has noted, however, that causes of death should be reported by medical examiners "in their own words" on the basis of clinical judgment, without allowing lists of conditions "to influence the reporting of any of the direct or antecedent causes of death."[28]

Excited delirium, while not generally recognized as a diagnostic term, is none-the-less commonly used in forensic science settings. In some localities, although the term "excited delirium" is not listed as a cause of death, characteristics of the syndrome may be listed as contributing factors.[29]

3. Physiological changes during ECD applications and excited delirium

3.1. Overview

Some of the factors mentioned in the following sections are interrelated, but are listed separately for discussion purposes. It would be unethical to apply extreme (i.e., long-duration or repeated) exposures of ECDs, which may cause harm or death, to human subjects. Therefore, some of the data discussed in this review are from studies of animals.

The first study of blood factors after any type of ECD exposure was performed by Jauchem et al.)[30] The swine as an animal model was selected for several reasons, including similarities to humans in terms of chemical and physical characteristics of blood, respiratory parameters, and responses to muscular exercise (for previous discussion, see Jauchem et al.), [4,30] and the ability to use animals in a weight range similar to that of potential human subjects. Tchou[31] commented that, regarding ECD studies, "pig model. . .findings have extrapolated well to human beings." Some experimentation, using ECDs, has been accomplished recently with human volunteers as subjects (studies described below).

An initial report of an animal model of a physiological state similar to excited delirium, for use in studies of ECD effects, was presented by Esquivel and Bir.[32] Although only preliminary data were reported, this innovative and unique model could prove to be very valuable in future studies. It would be unethical to administer drugs to human subjects at doses high enough or for long enough periods to simulate "street use" of such agents with subsequent induction of excited delirium. For these reasons (unlike the situation of ECD applications alone) there will be a paucity of relevant data presented in the sections below, relating to the syndrome.

3.2. Changes in blood potassium

Jauchem et al.[4,30,33] reported significant increases in blood potassium concentration ($[K^+]$) after ECD applications. It is well documented that the potassium efflux from contracting muscles stops at the end of exercise, while lactate continues to accumulate in extracellular fluids.[34] The time courses of blood $[K^+]$ and lactate results of Jauchem et al.[4,30,33] (see discussion of lactate in Section 3.7. *"Acidosis and blood lactate"* below) were consistent with this relationship; thus ECD applications, regarding these factors, are similar to exercise.

Maximal exercise in humans for 6 min can result in an increase in blood $[K^+]$ from, for example, 4.3 to 6.8 mEq/L.[35] The increases in the majority of studies of anesthetized swine, after ECD applications, were much less than that.[4,30] With more severe exposures,[33] blood $[K^+]$ reached as high as 7.6 mEq/L. "Severe hyperkalaemia" is generally defined as a blood $[K^+]$ of greater than 8 mEq/L.[36] It has been uniformly accepted that a serum $[K^+]$ of greater than 10 mEq/L is fatal unless urgent treatment is instituted)[37] (although one case of non-fatal hyperkalaemia was reported with a level of 14 mEq/L).[37] On the basis of Jauchem et al.'s anesthetized swine studies, there may be a wide margin of safety, relative to blood $[K^+]$, for most ECD applications.

Although reported cases of excited delirium, *per se*, have not included hyperkalaemia, rhabdomyolysis (including an increase in blood $[K^+]$) may be associated with cocaine use[38] (see Section 4 *"Confounding factors that may be present during ECD application and cases of excited delirium"* below). Rather than cocaine itself, however, adulterants used to "cut" the drug may be a cause of rhabdomyolysis.[39]

3.3. Effects on the cardiovascular system

Investigators have reported no ventricular fibrillation associated with waveforms matching the TASER X26 ECD (e.g., McDaniel et al.), [40] even with the probes positioned in close proximity along the cardiac axis.[41] Wu et al.,[42] on the basis of experimentation using swine, estimated an extremely low likelihood of ECD darts landing in a manner to cause ventricular fibrillation in humans (probability of 0.000172). Although Nanthakumar et al.[43] reported one case of ventricular fibrillation in an anesthetized swine that received epinephrine intravenously and was then exposed to an ECD, Kroll et al.[44] suggested that no objectively documented cases of ventricular fibrillation had occurred in 610,000 incidents of ECD use on humans during law-enforcement activities.

Nanthakumar et al.[43] reported "myocardial stimulation" due to TASER M26 and X26 ECDs applied to anesthetized swine. The definition of such "stimulation," however, was "change in electrogram morphology and rate during the discharge and perturbation of arterial blood pressure." These criteria would not necessarily imply any *direct* associated effects of the ECDs specifically on the heart. On the basis of modeling, Holden et al.[45] and Sun and Webster[46] concluded that current densities generated by an ECD would be extremely low, and that ventricular fibrillation was highly unlikely to occur as a result of any direct effect of ECD pulses on the heart. Cao et al.[47] also reported that a TASER X26 ECD application to a man, who had an implanted pacemaker, presumably caused "ventricular myocardial capture" (retrospectively determined by reviewing data storage in the pacemaker). The authors suggested that a preferential electrical pathway might have existed due to presence of

the pacemaker. It is possible, however, that there was no direct association between the medical device and the "high ventricular rate episodes corresponding to the exact time of the Taser application." Heart rate could have increased simply due to generalized stress.

In another study of swine by Dennis et al.,[48] two out of eight animals died due to ventricular fibrillation following two 40-s applications of a TASER X26 device. The animals in that study were anesthetized with ketamine/xylazine. Although ketamine/xylazine has been used in swine by other investigators, this combination may cause hypoxia due to hypoventilation.[49] Cardiac output and arterial pO_2 in swine are also significantly decreased after administration of ketamine/xylazine.[50] Ketamine/xylazine has been associated with some risk of respiratory arrest;[51] ketamine alone has been associated with a high incidence of arterial desaturation.[52] In one series of experiments, swine anesthetized with ketamine (versus other anesthetics) exhibited a higher incidence of ventricular fibrillation.[53] Whether these effects of ketamine or xylazine explain, in part, the deaths in the series of Dennis et al.[48] is unknown.

McDaniel et al.[40] reported only minor fluctuations in blood pressure of swine exposed to an ECD. The authors hypothesized that the fluctuations were caused by mechanical contractions of skeletal muscles, with no cardiac involvement. Vilke et al.[54] reported a significant *decrease* in systolic blood pressure in humans after exposure to the TASER X26 ECD. This difference was attributed to a "state of anxiety prior" to exposure. Thus simply the anticipation of the ECD application appeared to be an important factor.

Although the output of ECDs would not appear to be a common cause of ventricular fibrillation, other events common to the use of ECDs, such as ingestion of cocaine[38] may be associated with subsequent ventricular fibrillation. It is likely that cocaine does not cause excited delirium unless the heart and brain are already abnormal, or if the drug has been adulterated with a cardiotoxic agent. Victims of excited delirium generally have enlarged hearts[12,15,55] and exhibit asystole.[12] Cocaine does not cause ventricular fibrillation unless there is extensive underlying coronary artery disease. The association of chronic cocaine use with heart pathology was elucidated previously (e.g., by Tazelaar et al., [56] Karch and Billingham,[57] and Karch and Stephens[58]), and was recently reviewed by Karch.[59] Karch et al.[60] found that long-term cocaine use was necessary for the development of cardiac alterations, and that isolated postmortem measurements of blood cocaine concentrations (which may be high in occasional recreational users) should not be used to assess toxicity. In addition, Karch and Wetli[61] stressed that myocardial fibrosis and hypertrophy are common in general; both may promote arrhythmic sudden death. Anatomic alterations such as these may be substantial enough to cause death even without other events (including ECD applications) occurring.

One mechanism of death associated with excited delirium has been postulated to involve the sympathetic nervous system.[19] Struggling during an episode of the syndrome or the use of stimulant drugs (e.g., methamphetamine and cocaine) may activate the sympathetic nervous system, resulting in release of epinephrine and norepinephrine. Gulino and Young[62] suggested that deaths reported to be caused by "restraint asphyxia" were "most likely accounted for by catecholamine-mediated cardiac dysrhythmias." In addition, Fineschi et al.[63] noted an association of chronic hyperadrenergic states with disarray of cardiovascular ultrastructure.

3.4. Lack of hyperthermia during ECD applications

Human subjects and animals exposed to ECDs have not exhibited hyperthermia. Dawes et al.[64] reported that a 15-s ECD exposure did not cause core temperature elevation in resting adults. Jauchem et al.[33] found no change in rectal temperature even after many repeated ECD exposures in anesthetized swine.

Subjects who die after cocaine-associated excited delirium are often hyperthermic.[14] Karch,[15] however, noted that hyperthermia might not be present in all cases. He hypothesized that the hyperthermia is due to an imbalance between dopamine D1 and D2 receptors and that, depending on the length of drug use and underlying disease, the receptors may not yet be out of balance.

3.5. Respiration

Jauchem et al.[30] reported a lack of breathing during muscle contractions caused by experimental ECD applications in anesthetized swine. In experiments of extreme repeated ECD exposures,[33] respiratory arrest was the lethal endpoint in animals that did not survive. (The heart continued to exhibit organized QRS electrical activity after exposure. No ventricular fibrillation was observed). Esquivel et al.[65] noted impaired ventilation in anesthetized swine exposed to a different ECD. Although decreased respiration can be produced in animals under some experimental conditions, however, the validity of translating these results to human subjects is unknown. In contrast with the anesthetized swine experiments, Ho et al.[66,67] found that conscious humans were able to breathe during some ECD exposures, including those of relatively long duration.

While some investigators have suggested that death in excited delirium could be due to respiratory arrest (rather than cardiac arrest),[68] this has never been proven.

3.6. Rhabdomyolysis and muscle enzymes

Strong muscle contractions, such as those that may be present during ECD applications, could result in rhabdomyolysis and associated release of muscle enzymes into the bloodstream. Kroll[69] noted that TASER ECDs operate at a much lower pulse rate (19 pulses/s) than that which would cause full tetanus (therefore resulting in less potential muscle damage). Jauchem et al.[30] presented a graph of muscle contractions caused by the TASER X26 device, to illustrate the lack of full tetanus. In another study, after only three 5-s applications of the device, there was a statistically significant increase in serum myoglobin concentration.[4] The maximum value (a mean of 26 ng/ml in ten animals), however, was well below referenced upper limits of "normal" values for humans (which have included, e.g., 110 ng/ml).[70] Normal values for swine have not been defined. To place the degree of myoglobin increase in perspective, one should note that humans with exertional rhabdomyolysis have exhibited levels as high as 3750 ng/ml.[71] Since illicit drug use often occurs in conjunction with incidents involving TASER ECD use,[72] one should not assume that increases in blood myoglobin in such events are directly due to effects of the device.

Serum levels of cardiac troponin T and I (markers of cardiac muscle damage) were not significantly increased during a variety of ECD exposures of anesthetized swine.[4,30,33] In contrast, levels of cardiac troponin commonly increase (even as high as those that are characteristic of acute myocardial infarction) in runners who have, for example, just completed marathon races.[73] Any clinical significance of such an increase is unknown. Sloane et al.[74] found no abnormal levels of troponin I in human subjects six hr after receiving a single application of the TASER X26 ECD.

Rhabdomyolysis is a common consequence of illicit drug use.[75] Cocaine-induced rhabdomyolysis and excited delirium have been considered to be different stages of the same syndrome.[76] Rhabdomyolysis is almost always present in the rare patient who is resuscitated and dies of multisystem failure several days later,

presumably secondary to hyperthermia. Ordog et al.[77] reported mild rhabdomyolysis (and myoglobinuria) in 1% of a series of patients who had been exposed to a TASER ECD. Each of these patients, however, tested positive for phencyclidine. Thus, one could not determine whether muscle breakdown was due to the drug or to muscle contractions related to the ECD applications.

3.7. Acidosis and blood lactate

Blood pH was significantly decreased for one hr following 18 repeated TASER X26 device exposures, but subsequently returned toward a normal level.[30] Lactate was highly elevated, with a slow return toward baseline. In a subsequent study of only three repeated exposures of swine,[4] pH and lactate were significantly changed, but to a lesser degree than in the previous study. Valentino et al.[78] found only a moderate significant increase in blood lactate in swine (with no significant change in blood pH) due to applications of an ECD (manufactured by Aegis Industries, Bellevue, Idaho). It is not known, however, whether that particular ECD can cause incapacitating muscle contractions. In the experiments of Jauchem et al.,[4,30,33] blood lactate was the one factor that exhibited the greatest consistent change dependent on the relative amount of ECD exposure. Bozzuto[79] noted that physical exertion alone can result in severe hyperlactacidaemia.

Sloane and Vilke[80] suggested that any primary metabolic acidosis from excited delirium could be exacerbated by respiratory acidosis during ECD exposures. In addition, Manojlovic et al.[18] pointed out the potential synergistic impact of ECD exposures on pH in subjects with excited delirium. Even a single 5-s exposure to the X26 device has resulted in a significant increase in blood lactate in human subjects[66] (blood pH was not measured).

Because of the interruption of normal respiration during ECD applications in the anesthetized swine experiments, combined with extreme muscle contraction, one may use the term "respiratory acidosis with superimposed metabolic acidosis," as described by Brobst.[81] When considering repeated exposures to ECDs, a complex situation could result due to both muscle contractions and interruption of normal respiration. Both respiratory and metabolic changes could contribute to acidosis. In the experiments of Jauchem et al.,[4,30,33] even if respiratory effects could have been alleviated (e.g., by mechanical ventilation), it is likely that acidosis would have still been present, as reflected by the high levels of lactate. In addition, Tohdoh et al.[82] reported that excessive muscle contraction, in some instances, could induce both metabolic and respiratory acidosis.

Bouton et al.[83] noted, "Should TASER exposure cause acidosis, this might promote sudden death in subjects displaying symptoms of excited delirium." These authors found that a 5-s X26 ECD exposure resulted in a statistically significant decrease in blood pH (one min after exposure) and statistically significant increases in blood lactate (one and ten min after exposure).

One should not equate changes that may occur during chronic acid–base balance disturbances (e.g., ketoacidosis or lactic acidosis in chronic alcoholics or diabetics[84] with acute changes that may happen during and following ECD applications. In the experiments of Jauchem et al.,[4,30,33] pH returned to baseline levels relatively quickly.

4. Confounding factors that may be present during ECD application and cases of excited delirium

Active physical restraint and drug use are confounding factors that may occur during incidents involving either applications of ECDs or excited delirium. Law-enforcement personnel may utilize a "prone maximal restraint position" [85] to control combative subjects. In a series of 21 cases of death associated with excited delirium,[86] all of the subjects had undergone restraint. Chan et al.[87] concluded that such a position resulted in a "restrictive pulmonary function pattern," but with no evidence of hypoxia (i.e., no O_2 saturation less than 96%). This conclusion was based upon observations of normal subjects (not exhibiting excited delirium). Glatter and Karch[88] explained that "positional asphyxia," *per se*, could not result in any significant hypoxia. Since the body has massive O_2 reserves (maximum breathing capacity is about 50% greater than actual pulmonary ventilation), it is not possible to exhaust the amount of O_2 available under that condition. The term "positional asphyxia" may, in fact, only be appropriate for "intoxicated, massively obese individuals trapped in confined spaces," who are therefore unable to expand their chests.[15]

In a series of 18 cases of death associated with excited delirium and restraint,[89] each subject exhibited "labored or agonal breathing immediately before cardiopulmonary arrest." It is not uncommon for subjects with excited delirium to be observed by police officers as lacking respiratory activity (e.g., Lawrence).[90] During excited delirium, increased activity may also lead to an increase in O_2 demand.[91]

The potential enhancement of cocaine toxicity due to concurrent use of alcohol has been reviewed by Karch et al.,[92] who found little synergy between the two substances. There has been only one experimental study of humans who were purposely given alcohol and then exposed to ECDs.[93] Intoxicated subjects exhibited a small but statistically significant drop in pH (from 7.37 to 7.32) immediately following 15 s of ECD exposure. At 24-hour post-exposure, there was a small statistically significant increase in blood pCO_2 (46.3 vs. 42.4 mm Hg pre-exposure). There have been no experimental studies of humans involving administration of other drugs prior to ECD application.

Acute respiratory failure (including respiratory-muscle fatigue, which may be related to metabolic acidosis) due to substance abuse[94] may occur in subjects exhibiting excited delirium. Severe metabolic acidosis may be present at the time of death due to cocaine-induced excited delirium.[24] Cocaine use combined with exertion (as may occur during restraint of individuals during law-enforcement activities) can lead to a severe form of hyperlactacidaemia.[95] In one study of rhabdomyolysis, Gabow et al.[96] found that drugs and alcohol were responsible for 82% of all cases presented (see Section 3.6. "*Rhabdomyolysis and muscle enzymes*" above). It has not been demonstrated, however, that excited delirium, *per se*, causes rhabdomyolysis. To be more precise, people who arrest during the syndrome (and are resuscitated) usually develop rhabdomyolysis. Due to coexisting hyperthermia, it is not possible to attribute causation.

Stephens et al.[97] suggested, "Chronic drug use is necessary to induce the changes in the neurochemistry that lead to agitated delirium." Stephens et al.[98] described steps that should be considered early during an investigation whenever there is a basis to believe that cocaine is related to the cause of death. These steps would be particularly important during investigation of deaths in custody in which ECDs were involved. Other drugs, in addition to cocaine, found in the blood of subjects who died with presumed excited delirium, have been listed by Ross[13] and include: phencyclidine, methamphetamines, benzoylecgonine, lysergic acid, marijuana, lithium, methylphenidate HCl, haloperidol, lidocaine, and valproic acid. There is also a strong association of methamphetamine use with coronary artery disease and myocardial infarction.[99,100] The use of 3,4-methylenedioxymethamphetamine (MDMA) ("ecstasy") can result in fatal hyperthermia.[101] Some of these deaths can be partially due to behavior associated with use of the drug, such as protracted dancing without rest periods and lack of adequate fluid intake. There is evidence (both in animals and humans) that the drug causes peripheral vasoconstriction

Table 1
Evidence for association of factors with ECD applications or excited delirium

Factor	Electronic control devices	Excited delirium
Animal models	Most often anesthetized swine[4,30,31,33]	Anesthetized-swine model recently developed[32]
Hyperkalemia	Occurs even with low amount of exposure in animals,[4,30,33] but with wide margin of safety	May be caused by adulterants in drugs[39]
Ventricular fibrillation	Generally not linked,[40,41] and considered unlikely,[42] particularly as direct effect.[45,46] Cases reported in ketamine/xylazine-anesthetized animals.[48]	May be associated with drug use,[38] but possibly only with existing heart abnormalities.[12,15,55]
Hyperthermia	Not associated in animal[33] or human studies.[64]	Usually required for diagnosis;[14,15] often associated with cocaine.[14]
Respiratory effects	In anesthetized animals, lack of breathing during exposure;[4,30] respiratory arrest with extreme exposure.[33] Results not verified in humans[66,67]	Respiratory arrest required for diagnosis.[14,15]
Rhabdomyolysis, with release of muscle enzymes	↑ In blood myoglobin, even with low amount of exposure.[4] No abnormal levels of blood troponin in animals[4,30,33] nor in humans[74]	May be associated with drug use[75,76]
Acidosis	Significant ↓ in blood pH,[30,33] even with low amount of exposure.[4] Significant ↑ in blood lactate[66,83] and significant ↓ in blood pH[83] with single 5-s exposure in humans	Primary metabolic acidosis.[80] Could be associated with cocaine[24]
Listed as cause of death	Has been listed by some medical examiners,[10] despite lack of clear-cut unifying pathophysiological hypothesis	Death is usually required for diagnosis,[14,15] although some use the term "excited delirium" in cases with survival.[18] May be listed as "contributing factor"[29]

Superscript numerals refer to references in text.

and interferes with heat dissipation. In one study, 43% of all patients with a diagnosis of rhabdomyolysis tested positive for methamphetamine.[102]

Neurochemical changes associated with various drugs of abuse have been reviewed in a book edited by Karch.[103] Findings in certain chapters of the book, including discussion of postmortem neurochemical changes, should be of particular interest to forensic pathologists. For example, an increase in radioligand binding to dopaminergic transporters occurs in the brains of chronic cocaine users (without excited delirium), but not in victims of excited delirium.[104] Also, on the basis of postmortem analyses of human brains, changes in different dopamine receptor subtypes have been noted (with increases in some and decreases in others).[105] Although a number of postmortem changes in serotonin receptor subtypes have been identified in animals that received MDMA, more research is needed to verify these changes in human brains.[106] In addition to effects on the brain, Karch[107] hypothesized that misusers of methamphetamine may gain some level of myocardial protection (compared with cocaine misusers) due to increased amounts of inducible heat-shock proteins.

Channa Perera and Pollanen[108] noted, "It is important for the forensic pathologist to remember that death may occur suddenly during restraint from an unexpected mechanism other than excited delirium leading to cardiac arrhythmia or restraint asphyxia." The authors illustrated this by presenting a case of vaso-occlusive sickle cell crisis.

A summary of evidence for association of certain factors (mentioned in the sections above) with either ECD applications or excited delirium is presented in Table 1.

5. Suggestions by other investigators for use of ECDs when excited delirium may be present

The use of ECDs in mental health settings (with individuals experiencing a crisis) is controversial.[109] Some investigators have recommended enactment of policies prohibiting the "knowing use" of ECDs on mentally ill individuals (e.g., Stanford Criminal Justice Center).[110] Ho et al.,[111] however, found that, in nearly half of the cases in one study population, an ECD "was used in place of a firearm even though the law-enforcement use of deadly force would have been authorized and justified in these situations." On the other hand, Erwin and Philibert[112] questioned the ethics regarding any use of ECDs on psychiatric patients.

6. Concluding remarks

Primary effects of factors coincident with ECD exposure events may, by themselves, be more harmful than effects of limited ECD applications. Many victims of drug-induced excited delirium die without the application of any specific law-enforcement techniques.[14] Stone[113] noted that, on the basis of medical evidence, ECDs are not "the causes, in and of themselves," of sudden deaths in custody.

Some investigators have suggested that any description of ECD applications as "the last straw" or "being pushed over the edge," in cases of death during police confrontation, are simply "unscientific emotional explanations."[5] Others, however (e.g., Miller),[114] have raised concerns about subjects exhibiting excited delirium who would then subsequently be exposed to ECDs. Jenkinson et al.[3] noted, despite concerns about the medical aspects of ECD applications, "No police use-of-force option is risk-free." Drugs, physical exertion, and restraint may be more important factors leading to death than several short exposures to ECDs.[66]

DiMaio and Dana[19] stated that deaths following the use of ECDs "appear to be deaths due to excited delirium syndrome in individuals who coincidentally received Taser shocks." On the basis of the available literature cited in this current review, there could be more deaths from excited delirium alone than from ECD exposure alone (if, in fact, there are any from the latter). In a prospective multi-center cohort study of 962 subjects exposed to ECDs, two deaths in custody occurred; both were determined to be unrelated to ECD use.[115]

The occurrence of some statistically significant effects after some types of ECD applications should not be surprising. The role of such applications in deaths in custody, however, may be minor when compared with common aspects of excited delirium. Any risk/benefit analyses of the use of ECDs should include consideration of this point. As Drummer et al.[116] noted, "All aspects of the medicolegal death investigation triad – investigation (history), pathology, and laboratory results – are essential and must be evaluated in context with one another".

Conflict of interest

The author has not had any relationship with any manufacturers of electronic control devices, including employment, consultan-

cies, stock ownership, honoraria, paid expert testimony, patent applications/registrations, and grants or other funding.

Acknowledgements

This review was supported by the Directed Energy Research Programs Department, National Institute of Justice, Office of Justice Programs, United States Department of Justice.

The views and opinions expressed in this article are the author's own and do not necessarily state or reflect those of the US Government.

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